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CASE REPORT

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The term cotwin with spatial pulmonary lesions and elevated maternal-neonatal D-dimer levels following single intrauterine fetal death in a monochorionic-monoamniotic twin pregnancy: a case report and literature review

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Abstract

Background Complications such as cerebral impairment, preterm delivery, and even intrauterine death can occur in monochorionic twin pregnancies with singleton fetal death. The coagulation functions of the surviving fetus and mother remain controversial.

Case presentation We reported a case of spontaneous single intrauterine fetal death at 17 weeks of gestation in a monochorionic monoamniotic twin pregnancy, followed by increased maternal and neonatal D-dimer levels and spatial pulmonary lesions in the surviving term cotwin, without cerebral impairment. The mother experienced significant postpartum pelvic hematoma, which was managed with conservative treatment and resolved.

Conclusions Monitoring maternal and neonatal D-dimer, along with a detailed examination of the respiratory system and brain impairment of the surviving cotwin and newborn, maybe is crucial. This could be especially relevant in monochorionic monoamniotic twin pregnancies complicated by placental arterio-arterial or veno-venous anastomoses and elevated maternal D-dimer levels.

Keywords Monochorionic monoamniotic twin pregnancy, Spontaneous single intrauterine fetal death, D-dimer, Pulmonary lesion, Placental anastomoses

Introduction

Single intrauterine fetal death (sIUFD) occurs in 0.5–6.8% of all twin pregnancies [1–3], with the majority of fetal deaths occurring in the first trimester, posing a relatively low risk to the surviving cotwin [4]. However, in monochorionic twin pregnancies, sIUFD in the second or third trimester carries a significant risk for the surviving cotwin, with approximately 30–50% of surviving cotwins at risk of death or severe neurological injury [5, 6]. Monochorionic monoamniotic (MCMA) twins account for about 5% of all monochorionic pregnancies and less than 1% of all twin pregnancies [7]. These pregnancies are associated with high rates of stillbirth and perinatal mortality reported in the literature [8]. Here, we present a unique case of a surviving term cotwin with spatial pulmonary injury and transient elevated D-dimer levels but without brain impairment in an MCMA twin pregnancy complicated by spontaneous sIUFD at 17 weeks of gestation. We also conducted a review of monochorionic twin

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pregnancies complicated by sIUFD in the second or third trimester, excluding cases due to twin-twin transfusion syndrome (TTTS) or selected fetal growth restriction (sFGR).

Case report

A 39-year-old woman, gravida 3, para 2, underwent in vitro fertilization and embryo transfer, resulting in an MCMA twin pregnancy. Non-invasive DNA testing at 13 weeks of gestation indicated a low risk for trisomies 21, 18, and 13, with no other chromosomal or genomic screening conducted. At 17 weeks of gestation, one fetus was diagnosed with intrauterine death, and the maternal serum D-dimer level increased to 9.2 μg/mL. The mother had been asymptomatic since the previous normal scan at 15 weeks, with no signs of discordant fetal growth. She had a body mass index of 25.3, denied smoking and alcohol consumption, and had no significant medical history. The patient was started on subcutaneous nadroparin calcium at a dose of 4,200 U once a day, which continued until 38 weeks and 2 days of gestation. Maternal D-dimer levels fluctuated between 4 μg/mL and 20.4 μg/mL, without any associated abnormalities in other coagulation parameters. Throughout the pregnancy, the mother remained asymptomatic, and compression venous ultrasonography showed no abnormalities. The surviving fetus had normal cranial imaging via neurosonography and magnetic resonance imaging (MRI), with normal peak systolic velocity (PSV) of the middle cerebral artery (MCA) throughout the pregnancy. Fetal structural screening ultrasound at 22 weeks did not indicate any abnormalities in the lungs. Subsequent routine prenatal ultrasounds did not include detailed lung examinations. Due to a previous cesarean section (CS), the patient underwent a CS delivery at 39 weeks. Preoperative blood tests revealed a hemoglobin level of 128 g/L, D-dimer level of 20.40 μg/mL, fibrinogen level of 3.6 g/L, and normal results for other blood tests.

Two days after the CS, the patient appeared anemic with a pulse rate of 120 bpm. Physical examination revealed good uterine contraction with minimal lochia, without abdominal tenderness or rebound tenderness. Blood tests showed a decrease in hemoglobin to 60 g/L, D-dimer to 4.96 μg/mL, and creatinine to 106 μmol/L. A pelvic ultrasound revealed a massive hematoma measuring 8 cm ×7 cm ×5 cm in the right lower abdomen. A blood transfusion was administered, and following supportive care, the maternal vital signs stabilized. The hematoma's size remained stable at 14 cm ×10 cm ×7 cm, and hemoglobin levels increased to 80 g/L with a serum creatinine level of 80 μmol/L. Reoperation was not considered.

The patient was given an external Chinese medicine hemostatic prescription for the hematoma, along with nadroparin calcium and medical compression stockings to prevent deep venous thrombosis (DVT). She was discharged 10 days after the CS, with a hemoglobin level of 92 g/L. Eight weeks later, a transvaginal ultrasound revealed a shrinking hematoma measuring 9.2 cm ×8.7 cm ×7.8 cm, and the D-dimer level returned to 2.24 μg/mL. The fluctuations in D-dimer levels are shown in Fig. 1.

After birth, the infant showed no clinical signs of neurological impairment, and brain MRI imaging was normal. However, the term infant exhibited dyspnea two hours after birth, and a chest X-ray revealed bilateral lungs exudation (Fig. 2A). After intubation and mechanical ventilation, the patient's dyspnea improved, and on day 4, ventilation was discontinued. Notably, routine blood tests for neutrophil and leukocyte counts, as well as infection markers such as C-reactive protein and serum amyloid A, were all within normal ranges. Negative blood, sputum, chlamydia, and mycoplasma cultures ruled out infection. The bilateral exudation seen on chest X-ray improved by day 10 (Fig. 2B). A coagulation test on the sixth day after birth showed an elevated D-dimer level of 29.88 μg/mL, though no other coagulation abnormalities or thromboembolic complications were observed. The D-dimer level returned to normal by day 8, and the infant was discharged on day 10. No neurological delays were observed in the neonate.

Written informed consent was obtained from the patients for the publication of all images, clinical data, and other data included in this manuscript.

Discussion

The occurrence of surviving cotwins born at term without brain injury following a second-trimester spontaneous sIUFD within a MCMA twin pregnancy is exceedingly rare. After sIUFD, maternal D-dimer levels exhibited a significant increase throughout the entire gestational period. Upon birth, the surviving full-term newborn demonstrated a transient elevation in D-dimer levels within the first 8 days, coinciding with unexplained noninflammatory exudative lesions observed in the bilateral pulmonary region. However, the coagulation status of the mother, surviving fetus, and newborn following sIUFD remains poorly understood in the literature. To address this knowledge gap, a comprehensive literature review was conducted, focusing on cases of monochorionic pregnancies complicated by spontaneous sIUFD in the second or third trimester, excluding those attributed to TTTS or sFGR. This review aimed to elucidate the clinical characteristics of such cases, identify potential risk factors for neonatal pulmonary lesions, and highlight

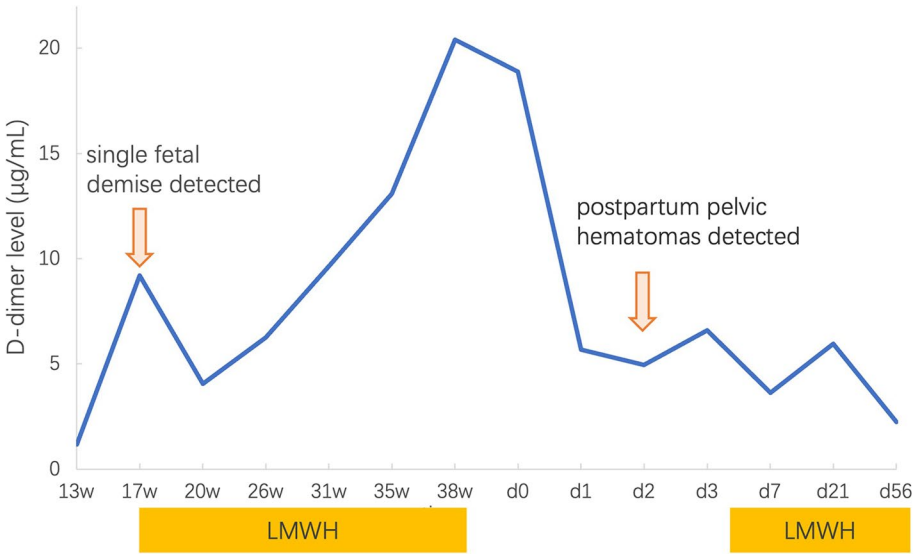

Fig. 1 Changes in D-dimer levels during pregnancy and puerperium. The maternal D-dimer level significantly increased to 9.2 μg/mL following single fetal death in a monochorionic-monoamniotic twin pregnancy at 17 weeks of gestation and decreased to 4 μg/mL at 20 weeks of gestation after the administration of nadroparin calcium. Then, the D-dimer level continuously increased to a maximum of 20.4 μg/mL at 38 weeks of gestation and returned to 2.24 mg/L at 8 weeks postpartum. 13 w, 13 weeks of gestation, and so on. d1, one day postpartum, and so on. LMWH, low molecular weight heparin

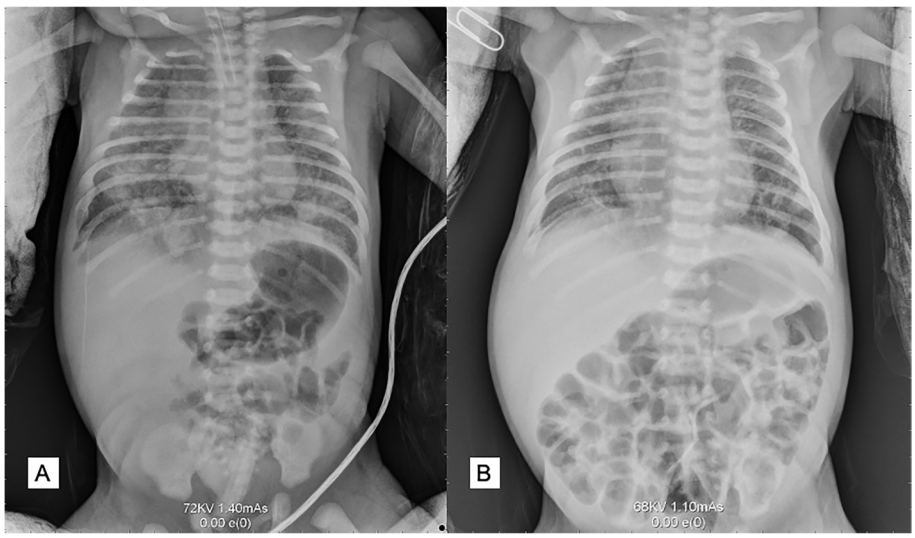

Fig. 2 Chest radiographs of the neonate. The exudative lesions in both lungs improved 10 days later. **A** Chest radiograph on the first day after birth. **B** Chest radiograph on the 10 th day after birth

the importance of prenatal assessments that include maternal and neonatal D-dimer levels and potential end-organ damage (e.g., the respiratory system) in surviving cotwins, particularly in MCMA pregnancies with elevated maternal D-dimer levels.

The cause of the single fetal death in this patient remains unknown. Upon examination at delivery, the deceased fetus was identified as a fetal papyraceus with an atrophic umbilical cord. The insertion points of the two umbilical cords were 1 cm apart, without evidence of velamentous insertion. In monoamniotic twin pregnancies, a significant proportion of fetal deaths are caused by fetal defects, while in structurally normal cases, death is often attributed to tight cord entanglement [9]. These factors may have contributed to the single fetal death in this patient.

In our review (Table 1), five surviving cotwins (7.6%, 5/66) experienced intrauterine death, 50 cotwins (82%, 50/61) were born prematurely, 14 neonates (23.0%, 14/61) died after birth, 26 neonates (44.1%, 26/59) had intracranial lesions, and 7 preterm newborns developed other organ impairments, including lung damage, kidney abnormalities, intestinal injury, and cardiac failure, primarily due to complications of prematurity. Additionally, brain MRI and neurosonography showed that the surviving fetus in this case had normal cranial imaging, even at term. Surviving twins in cases of single fetal death are at a high risk for abnormal postnatal brain imaging, especially in monochorionic pregnancies, with an incidence of approximately 43% [22], consistent with our reviewed case series.

Two main theories explain the increased morbidity and mortality risk for the surviving cotwin in monochorionic pregnancies: "twin embolization syndrome" and "hemodynamic imbalance." The hemodynamic imbalance theory, which posits that placental intertwin anastomoses allow the transfer of blood from the surviving cotwin to the deceased twin, causing periods of hypoperfusion and adverse outcomes such as neurological changes, is widely accepted. This theory is supported by evidence of fetal anemia in the surviving cotwin, as documented in fetal blood sampling [23–25]. Sonographic measurement of the MCA PSV is a reliable and noninvasive method to detect fetal anemia. Bajoria et al. established that superficial intertwin arterio-arterial or veno-venous anastomoses increase the incidence of intrauterine death, fetal anemia, and neurological impairments [15]. As shown in Table 1, of the 17 surviving fetuses with placental intertwin arterio-arterial or veno-venous anastomoses, 11 (64.7%, 11/17) developed anemia, and 10 of the 11 anemic fetuses (90.9%, 10/11) developed brain damage. In contrast, of the nine surviving fetuses with placental intertwin arterio-venous or veno-arterial vascular shunts, two (22.2%, 2/9) developed anemia followed by neurological impairment. The risk to the surviving monochorionic cotwin may depend on the type and size of the placental anastomoses. Cotwin anemia and even death can result from acute hemodynamic imbalances caused by large placental anastomoses, such as arterio-arterial and veno-venous anastomoses. Fortunately, the MCA PSV of the surviving cotwin in our patient was normal, indicating that our patient did not have large arterio-arterial or veno-venous anastomoses. The delineation of superficial placental anastomoses by power Doppler ultrasound or MRI has become possible in research and may help predict the prognosis of the surviving fetus, guiding clinical management.

Furthermore, the neonate in our case exhibited transient D-dimer elevation, with a maximum of 29.88 μg/mL, an extremely high level. However, previous reports indicate that coagulation screening tests on the surviving infant twin at delivery were almost always normal when complications of prematurity were excluded [26]. Khalilov et al. reported that the mean D-dimer level in healthy term neonates at one week (2.44 ± 2.45 μg/mL) was higher than that in adults (< 0.5 μg/mL), and D-dimer levels gradually decreased over the first month [27]. The high D-dimer levels in the neonatal period may be attributed to factors such as diminished renal clearance, birth stress, circulatory adaptation, short-term hypoxia, or intrauterine activation [27]. The transient elevation of D-dimer in the newborn in our case may have been triggered by intrauterine activation related to the deceased fetus. However, the gradient was insufficient to allow thromboembolic material to flow from the dead fetus to the circulation of the survivor. It is possible that other pathways activated the coagulation system in the surviving fetus.

In our patient, after 3 days of intubation and mechanical ventilation and 10 days of anti-inflammatory treatment with ampicillin sulbactam sodium and ceftazidime, the neonatal exudative lesions in both lungs, as well as other clinical symptoms, improved significantly. Neonatal wet lung disease is typically self-limiting, with complete remission within 2–3 days. Respiratory distress syndrome, a progressive condition frequently observed in preterm infants and mothers with diabetes, is characterized by reduced transparency and ground-glass opacities, and even white lungs on chest radiographs. Our case did not resemble either of these diseases. During the intrauterine period, the fetus lacks pulmonary circulation. Following birth, thrombotic material from the deceased fetus may enter the neonate's pulmonary circulation, causing pulmonary damage. Such lung damage is usually mild, and is not consistent with our case. However, in our case, both fetuses were housed within a single amniotic sac. Following the occurrence of sIUFD, necrotic material from the deceased fetus may have been released into the shared amniotic cavity. A small amount of amniotic fluid containing this necrotic material could have entered the lungs of the surviving fetus, potentially leading to obstruction and the development of exudative lesions. These lesions likely went undetected during routine prenatal ultrasounds. Upon delivery, the initiation of neonatal pulmonary circulation may have further exacerbated these lung lesions. This interpretation remains speculative, and further research is required to confirm this hypothesis. Szymonowicz W et al. reported three cases of pulmonary complications in monochorionic diamniotic twin pregnancies, including two cases of pulmonary infarction and one case of pulmonary artery embolism [11]. The gestational ages at delivery were 28, 31, and 32

Table 1 Reported spontaneous single intrauterine fetal death in monochorionic twin without twin-twin transfusion syndrome or selected fetal growth restriction [1, 3, 10–21]

Author	Number of cases	Mono or Di-amnioticity	GA at first sIUFD (weeks)	GA at second death/delivery (weeks)	Outcome of co-twin						Type of vascular shunts in placentas	Maternal coagulation function	Time interval (days)
					IUFD	Neonatal death	Anemia at first death	Intracranial lesions	other systemic abnormalities in the newborns	Well			
Romero R et al. 1984 [10]	1	diamniotic	26	36	0	0	NA	0	0	1	NA	decreased serum fibrinogen	70
Enbom JA et al. 1985 [1]	2	diamniotic	33/25	35/36.6	0	0	NA	0	0	2	NA	decreased serum fibrinogen / normal	14/81
Szymonowicz W et al. 1986 [11]	6	diamniotic	24.5(20.8, 29.5)	31.5(30.3, 33.5)	0	4	NA	6	2 cases with pulmonary infarction, spleen infarction and renal cortical necrosis or renal infarction, 1 case with pulmonary artery embolism and multiple infarcts in the liver	0	NA	NA	45.5(28.0, 61.3)
Fusi L et al. 1990 [12]	3	diamniotic	30/24/31.9	37/30/33	0	1	NA	1	1 cardiac failure	1	NA	normal	49/42/8
Gaucherand P et al. 1994 [13]	5	diamniotic	30.0(21.0, 34.0)	37.9(35.5, 38.6)	0	0	NA	1	0	1	NA	NA	49.0(7.0, 108.5)
Nicolini U et al. 1998 [14]	1	diamniotic	34	34	0	0	1	1	0	0	NA	NA	0
Bajoria R et al. 1999 [15]	15	diamniotic	28.2(27.1, 30.0)	28.4(27.1, 30.2)	4	8	10	9	0	1	AA/VV	NA	1.0 (0.5, 3.0)
Bajoria R et al. 1999 [15]	9	diamniotic	31.0(29.2, 36.0)	34.3(30.0, 38.0)	0	0	2 (NA 1)	2 (NA 2)	0	6 (NA 3)	AV/VA	NA	7.0 (1.8, 16.5)
Petersen I et al. 1999 [16]	1	diamniotic	30.6	32.6	0	0	NA	0	0	1	NA	normal	14
Saito K et al. 1999 [17]	2	monoamniotic	27.4/36	27.6/36	0	0	NA	1	0	1	NA	NA	1/0
Saito K et al. 1999 [17]	11	diamniotic	31.7(24.6, 33.6)	33.9(28.0, 35.7)	0	1	NA	1	0	7	NA	NA	14.0(3.0, 31.0)
Woo H et al. 2000 [3]	2	monoamniotic	19/35.7	30.9/35.7	1	0	NA	0	one with multiple congenital abnormalities	0	NA	NA	11.9/0
Woo H et al. 2000 [3]	3	diamniotic	35/35.9/37.1	35/35.9/37.1	0	0	NA	1	0	2	NA	NA	0
Miura N et al. 2008 [18]	1	diamniotic	21.7	28.7	0	0	0	0	0	1	AA	NA	49
Morokum S et al. 2008 [19]	1	diamniotic	18.9	41	0	0	NA	1	0	0	NA	NA	154
Machino H et al. 2017 [20]	1	monoamniotic	21	36	0	0	0	1	renal hypoplasia	0	NA	NA	105
Hui PW et al. 2021 [21]	1	diamniotic	15.6	34.3	0	0	1	1	intestinal atresia	0	AA	NA	61

Table 1 (continued)

Date was presented as (median, interquartile range) or (n, %)

siufd single intrauterine fetal death, *GA* Gestational age, *AA* Arterio-arterial,*VV* Veno-venous, *AV* Arterio-venous, *VA* veno-arterial,*time interval* time interval between first and second death and/or between first death and live birth of the co-twin,*NA* Not available

weeks, respectively. Therefore, lung damage due to complications of prematurity cannot be excluded. Notably, the surviving term twin in our case did not develop brain damage, although spatial pulmonary lesions were present postnatally. The possible risk factors for the pulmonary lesions in our patient include elevated maternal D-dimer levels and the MCMA pregnancy.

Maternal disseminated intravascular coagulation (DIC) is extremely rare in multiple pregnancies following sIUFD [28, 29], although the cause remains unclear. Maternal coagulation function results were available for only eight patients, with two showing decreased fibrinogen levels and five showing normal fibrinogen levels. Only our patient exhibited an increase in maternal D-dimer levels, from 1.18 μg/mL to 9.21 μg/mL following single fetal demise at 17 weeks of gestation, indicating activation of maternal fibrinolysis. The D-dimer concentration decreased to 4.05 μg/mL at 20 weeks of gestation, possibly due to self-regulation of the maternal coagulation-fibrinolysis system and the effect of low-molecular-weight heparin (LMWH). D-dimer levels are useful markers for early diagnosis of DIC and thromboembolism [30, 31]. However, there were no clinical manifestations of thromboembolic complications in this mother. Small emboli in the deep pelvic veins cannot be ruled out, as pelvic DVT is difficult to diagnose during pregnancy. Even if present, the emboli were likely small and had minimal impact. Similarly, Daniilidis et al. [32] reported elevated D-dimer levels, with a maximum of approximately 10 μg/mL at 33 weeks of gestation following a single twin death, without the occurrence of DIC or thromboembolism in the mother. Therefore, maybe it is advisable to assess the baseline coagulation profile, including D-dimer levels, when sIUFD occurs.

D-dimer, a fibrinolytic-specific degradation product, is a highly sensitive marker of fibrin formation. Elevated D-dimer concentrations indicate activation of both the coagulation and fibrinolytic systems. During normal pregnancy, D-dimer levels gradually increase, reaching a peak in the third trimester and remaining elevated for up to 48 h postpartum. This elevation is due to the increased demand for fibrin formation and fibrinolysis, which helps maintain adequate blood flow to the placenta and fetus while reducing the risk of postpartum bleeding [33–37]. Previous studies have demonstrated that elevated maternal D-dimer levels are independent risk factors for postpartum hemorrhage [34, 38]. In the case of our patient, severe pelvic bleeding occurred two days postpartum. Although the possibility of this pelvic hematoma as a surgical complication following the cesarean section could not be entirely excluded, it is important to note that the two surgeons performing the procedure were senior obstetric directors, and the surgery itself proceeded without incident. As such, it was deemed relatively unlikely that the pelvic hematoma was a result of a surgical complication. Instead, the bleeding may be attributed to the delivery process, combined with increased fibrinolysis as indicated by elevated D-dimer levels. The consumption of fibrinogen leads to relatively low fibrinogen levels, which, in turn, increases the risk of postpartum hemorrhage. The pathophysiology behind D-dimer elevation and its sustained effects on maternal and fetal outcomes, especially its association with maternal pelvic hemorrhage and neonatal pulmonary lesions, require further investigation through clinical and laboratory studies.

In this case, following a single fetal death and an increase in maternal D-dimer, LMWH was administered until 38 weeks and 2 days of gestation. During this period, D-dimer levels fluctuated at relatively high levels. Fortunately, no cerebral impairment was noted in the neonate. However, two days postpartum, pelvic hemorrhage occurred, and the newborn developed unexplained pulmonary lesions, with transient elevated D-dimer levels. The potential relationship between these complications and LMWH use remains unclear, although the administration of LMWH during the prenatal period is generally considered safe.

MCMA twin pregnancies are always accompanied by multiple complications. Therefore, the diagnosis of chorionicity and amnionicity during the first trimester is essential for optimal pregnancy management and follow-up [39]. After the first trimester, we typically perform ultrasounds every two weeks to monitor fetal viability, assess for growth restriction, and screen for conditions such as TTTS, and twin anemia-polycythemia sequences in monochorionic pregnancies. Upon detection of a single intrauterine fetal death, the MCA PSV of the surviving cotwin and maternal coagulation function, including D-dimer levels, are assessed immediately. If abnormal coagulation is detected, regular follow-up and LMWH administration are considered when necessary. Cranial imaging of the surviving cotwin is conducted using MRI three weeks later, and further follow-up is performed through MRI and fetal neurosonography throughout the pregnancy. Ideally, it would be beneficial to assess the

type and size of placental intertwin anastomoses using MRI or ultrasound.

Conclusions

In addition to assessing the brain impairment of the surviving cotwin in cases of sIUFD, careful evaluation of potential damage to other end organs, such as the respiratory system, maybe is also necessary. This can be particularly important in MCMA pregnancies complicated by placental arterio-arterial or veno-venous anastomoses and elevated maternal D-dimer levels. Furthermore, elevated maternal D-dimer levels may necessitate closely monitoring of both maternal and neonatal coagulation indicators, including D-dimer, throughout the course of care.

Abbreviations

sIUFD	Single intrauterine fetal death
MCMA	Monochorionic monoamniotic
TTTS	Twin-twin transfusion syndrome
sFGR	Selected fetal growth restriction
CS	Cesarean section
CRP	C-reactive protein
WBC	White blood cell
FDP	Fibrinogen degradation products
MCA	The middle cerebral artery
PSV	Peak systolic velocity
DVT	Deep venous thrombosis
DIC	Disseminated intravascular coagulation
LMWH	Low molecular weight heparin
MRI	Magnetic resonance imaging

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Authors' contributions

Haiyan Liu participated in patient management, conceived and designed the study, and was the principal contributor to writing the manuscript. Xiaoyue Zhang acquired, analyzed, and interpreted the data, and wrote and revised the manuscript. Zhenzhen Liu and Yi Yu participated in patient management and analysis of patient data. Weirong Gu participated in patient management, conceived and designed the study, and was a major contributor to revising the manuscript. All authors have read and approved the final manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The study was approved by the Institutional Ethics Committee of our hospital, and written informed consent was obtained from the patient.

Consent for publication

Written informed consent for the publication of clinical details and clinical images was obtained from the patient and the patient's guardian. A copy of the consent form is available for review by the Editor of this journal.

Competing interests

The authors declare no competing interests.

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